South pacific underwater medicine society

Journal/*Newsletter*

C O N T E N T S

SPUMS JOURNAL/NEWSLETTER

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Editorial

Diving is full of paradoxes, not the least being that while it is self evident that submergence in a non-life-supporting fluid should be dangerous yet fatalities are surprisingly few, and those that occur could usually have been avoided by the application of present advised practices. Notwithstanding this there is much that is still only partially understood, or hotly disputed, concerning present problems and a strong presumption that additional even more limiting difficulties will be recognised as diving continues to go deeper and longer on non-air. No clues as to either unsuspected problems or to possible answers can be ignored and this is the reason for the use in these columns of articles not overly medical in type. The "Business" side of diving is significant in its effect on diving practices and the requirements divers are exhorted to meet. At one time all roads were said to lead to Rome, so now all diving problems are ultimately solved by Diving Medicine (if solved is the correct word). The diver is our "patient" and his healthy survival our concern, especially so if we are the diver.

The notice of the Insurance requirements for those attending the RAN courses highlights the question of whether diving is, on Insurance terms, risky. Certainly the Insurance Companies do NOT know, they merely guess so: and the RAN demand for a $150,000 "cover" for each of the few attendees implies that either they are considered a major risk to the RAN or that the RAN diving organisation is dangerous. Neither seems likely, but the very lack of reliable information concerning actual diving morbidity and mortality risks makes it difficult to counter such suggestions. Diligent readers may have noticed subtle references to the need for Incident Reports appear from time to time in these pages, and this is as good a reason as any for another appeal.

The Melbourne meeting seems to have been favoured with papers, any one of which could be taken as the theme for a full Workshop discussion. It is hoped that more information will appear in future issues concerning these presentations. The mention by Dr Malcolm Evans of Keatinge's work on the value of small quantities of alcohol on survival brings back to mind an occasion many years ago when I tried to follow up a report that German aircrew who ditched in the Channel survived better than did Allied crews. The Germans were said to be issued with spirits in their survival packs. Unfortunately the German records had been destroyed in the War so I wrote to the MRC in UK. They told me that as everyone knew alcohol was deleterious to survival they were rather surprised that I raised the matter. Squashed I might be, but respectful of their Curiosity Index I was not! It is very difficult to recognise facts that fail to fit into one's knowledge of what really is the truth. As Thurber once said, it is better to ask some of the questions than to know all the answers. In this respect Diving Medicine is stimulating, so much remaining still debatable. The discussion on Decompression Sickness at Melbourne seems to demonstrate that the only area of total agreement is that divers make bad yo-yos and a little surface oxygen passes the time safely while you work out your next move. Predictable also was the raising of metaphorical hackles over the use of one-man chambers for the treatment of Bends, the old question of best practicable versus best possible. And the DCM argument seems likely to continue until a better model of diver is produced, for the machines can do all the diving anyone could desire and never once fall sick. As for Bob Montgomery's paper, he apparently only omitted one thing; he didn't say whether wives are right in thinking that all divers must be mad.

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SUBSCRIPTIONS

Members pay $15 yearly. Associate membership for those neither medically qualified nor engaged in hyperbaric nor underwater related research is available for $10. The journal is sent up to four issues yearly to both full and associate members. Those resident outside the immediate Australasian area should write for the special terms available.

Treasurer: Mr W Rehfisch, 5 Allawah Avenue, Frankston, VIC 3199.

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Notes to Correspondents and Authors

Please type all correspondence and be certain to give your name and address even though they may not be for publication. Authors are requested to be considerate of the limited facilities for the redrawing of tables, graphs or illustrations and should provide same in a presentation suitable for photo-reproduction direct. Books, journals, notices of Symposia, etc will be given consideration for notice in this journal.

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Disclaimer

All opinions expressed are given in good faith and in all cases represent the views of the writer and are not necessarily representative of the policy of SPUMS.

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DIVING COURSES

Courses in Underwater Medicine at the Royal Australian Navy School of Underwater Medicine, HMAS PENGUIN.

The Royal Australian Navy is willing to accept up to five civilian members of the South Pacific Underwater Medicine Society on each course conducted at the School of Underwater Medicine. The actual number of places available will depend on Service medical officer enrolments.

The Course for 1977 will be:

Medical Officers' Preliminary Course 7th to 18th March
Medical Officers' Advanced Course 21st March to 1st of April
Medical Officers' Preliminary Course 7th to 18th November.

Mid-day meals will be available on a repayment basis, but no accommodation will be available in HMAS PENGUIN.

Those attending courses will have to sign a form of indemnity releasing the Commonwealth of Australia from all responsibility (this form will be provided by the Society) and will have to produce proof of personal accident insurance cover in respect of individual members and the Society in respect of injury or damage however caused to either the insured, the Commonwealth or others whilst on course in the sum of $150,000 any one injury, unlimited in all. The Society is investigating the cost of such insurance.

Any members wishing to be sponsored for these courses should write to the Secretary (Dr John Knight, 80 Wellington Parade, East Melbourne 3002) at least 8 weeks before the course is due to commence. Applicants should state their full names, address, date and place of birth, dates and details of medical qualifications, diving experience and reasons for requiring the course. This information will be forwarded to the Royal Australian Navy. After some weeks confirmation of acceptance and detailed joining instructions will be issued. Early application is strongly advised to allow time for administrative delays.

Only those who have completed the Preliminary Course will be allowed to proceed to the Advanced Course. Courses may be taken in different years at times suitable to the candidate. These courses are essential for anyone contemplating sitting for the Diploma of Diving and Hyperbaric Medicine.

It is intended to appoint a Diploma Secretary who will be resident in Sydney and who will be responsible for the Society's side of the administration of these courses and of the course in Hyperbaric Medicine at Prince Henry Hospital, Little Bay which is also a required course for the Diploma.

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In-water Oxygen therapy can be limited to 10m by so limiting the gas supply hose.

A very lucky diver

Keith Momery, a 23 year old skin-diver, became unconscious and sank while diving off Penzance, UK, recently. His life was saved through the action of Beaky, a dolphin well known to the local divers, who swam down and not only brought him to the surface but kept him afloat till rescuers arrived. Not everyone who dives alone can expect such providential succour.

SPUMS MELBOURNE MEETING OCTOBER, 1976

The scientific meeting was held in the Clinical Lecture Theatre at Prince Henry's Hospital, St Kilda Road, Melbourne on Saturday morning October 30th. In all 32 people, mostly members of SPUMS, attended and apologies were received from a further half dozen members.

The meeting opened with a few words from the President, Dr Ian Unsworth, who showed two very interesting slides of the behaviour of decompression meters subjected to air dives in a chamber. They indicated approximately the correct profile, for USN decompression tables, for the first dive, and were even a little cautious on the longer dive. But the second and subsequent dives the meters failed completely to indicate reasonable decompressions. In fact for repeated deep short dives the meters failed completely to allow for the previous dives. As Dr Unsworth said, to follow the meter was a good way to acquire decompression sickness.

The first speaker was Dr Malcolm Evans, who first had experience of his subject while in the Royal Navy. He has been diving with the RN, has served with the Falkland Islands Dependencies Survey, where they chat up the penguins every winter, and worked with Dr Keatinge in studying the effects of cold on immersed humans. He spoke on "Cold and the Diver". He described the effects of immersion on the body and the effects of sudden immersion in cold water and the adjustments that thinking man can make to survive in a cold environment. One of the interesting sidelights was that the ingestion of alcohol, not to the stage of intoxication, improved tolerance, in the conditions of the Cambridge experiments, to cold and improved performance. The post-alcohol subjects were able to pull themselves from the water after an exposure which, without alcohol, they had had to be lifted out. There does not seem to be an explanation for this finding.

The Royal Naval film "Decompression Sickness, Part 1 - Bending" was shown. It is good training film explaining the current concepts of the causation of decompression sickness clearly. It has been metricated. It covers the theory of increased inert gas dissolving with increased pressure, and the outline of the theory, but not the detail, of decompression at the end of a dive and of therapeutic decompression. It emphasises the need to follow decompression tables.

Then Dr Geoff Macfarlane spoke on the "Clinical Presentation of Decompression Sickness", and gave some interesting case histories. He believes in treating patients with decompression sickness with hyperbaric oxygen to the point of toxicity, in replacing the large volume deficits that seriously ill patients have, and preventing intravascular coagulopathy with small doses of heparin.

Dr Gavin Dawson, of Prince Henry's Hospital, then described the hospital's one-man Vickers hyperbaric unit and weighed up very favourably the pros and cons of such a unit for the treatment of decompression sickness. The main advantages being that there is an oxygen atmosphere and so there is no doubt about the mixture being breathed and there is an extra escape route for nitrogen through the skin, the patient is being denitrogenated as effectively as possible, and that the attendant is not exposed to a hyperbaric environment. Against this there is no way, except by decompressing, that the patient can be reached should anything go wrong. It is very difficult to maintain a drip. However there is now a fluidic controlled ventilator which has been used in the chamber for artificial ventilation.

Dr Arthur Keech, the Medical Officer to the Melbourne Metropolitan Board of Works, spoke on the facilities for treatment owned by the Board. These are provided for the treatment of compressed air workers who are affected by decompression sickness.

He gave two case histories which illustrated the difficulties of therapy in decompression sickness.

Surgeon Commander Geoff Bayliss, RAN, spoke of the facilities available in various situations and took the meeting to HMS RECLAIM in Lock Fyne, a big research deep diving facility in the USA, showed the one man surface decompression chamber, which should never be used for therapeutic purposes as it is air pressurised and the patient is completely inaccessible, and almost invisible. He recommended immediate compression to 165 feet and then holding the patient at that depth while getting expert advice. He warned against the practice of yo-yoing, failing to relieve the patient's symptoms completely and then reducing pressure to see what would happen. What usually happens is that the patient gets worse and has to be recompressed. But now as a result of the bubble expansion he has a larger area or rather volume of damaged tissue round each bubble and is in worse condition than he was before.

Dr John Knight spoke on the treatments available if one did not have access to a recompression chamber. Oxygen at the surface, 100% by face mask would do no harm and might, in the less affected, relieve symptoms. Compression in water breathing oxygen (see Diving and Subaquatic Medicine, Edmonds, Lowry and Pennefather, 1976 p 389) could be considered if the equipment was available. The equipment, full facemask (Visionair) 12 meters of oxygen hose, large oxygen cylinder and reducing valve set at 100 lb/in^2, was demonstrated on Dr Phillip Rubinstein. A case history of successful treatment of neurological decompression sickness (Saumarez, Bolt & Gregory BMJ, i: 151, 1973) without the use of recompression was discussed.

At the end of the session on decompression sickness there were a number of questions and it became evident that the members of the panel did not always agree among themselves on what was the best treatment fer a particular problem. This is not surprising in a disease with as variable a presentation and clinical course as decompression sickness. All were agreed however that education of divers about the dangers of foolhardy decompression was very desirable.

Dr Robert Montgomery, a Psychologist from Latrobe University and a diver, spoke about the problems of diving from the psychological view point. He explained how the diver was insulated from many of the normal experiences of life when underwater: his wet suit cut out much of the skin stimulation, his mask cut down his visual field, water altered the auditory responses and all he heard most of the time were the bubbles bursting from his regulator. Into this understimulated environment came a problem and the sudden stimulus could overload his capacity to cope and then panic was close by and with panic came inappropriate actions. This short summary does less than justice to Bob ' s excellent presentation.

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Some people are abnormally sensitive to decompression sickness. One New Zealand diver is so liable that he must limit himself to 20 ft depth maximum.

Chest pain after a dive may indicate mediastinal emphysema or myocardial ischaemia.

Many divers are too buoyant to maintain a 10ft or 20ft decompression stop depth. Sport divers should avoid dives requiring decompression stops.

REPORT OF DIVING INCIDENT,

Dr DR Kerr, MBBS, Anaesthetics Registrar

On Thursday 29th January, my buddy and I went for a dive off Glacier Point. The dive lasted about one hour and maximum depth was probably 15 feet. Visibility was poor and diving the dive we surfaced 3-4 times to check our position. The purpose of the dive was also to examine a model of an underwater refuge I had been working on. This consisted of a large plastic bag tethered to a rock and filled with air from our exhale ports. At one stage this broke loose and in an attempt to prevent it surfacing I held onto it, but as I was ascending rapidly I let go after about 2 metres. We also used the bag to raise an old oil drum on the sea bed. This we also accompanied to the surface, but at standard ascent rates.

After the dive, all was well until about 24 hours later when, I developed a severe dull aching pain in my left shoulder, more in the axilla than on the shoulder tip. There was radiation down my arm and down both back and front of my chest. The pain came on over about half an hour and at first felt muscular in origin, but by the time it had developed fully I thought it was more likely to be a pneumothorax.

Symptoms began to subside after I arrived home and since I had no respiratory distress I decided to wait and see what developed. About 4.00 am I was wakened by very loud clicking sounds associated with each heartbeat. This was recognised as Hammans sign, diagnostic of pneumonediastinum. The sound was up to 10 feet away in a quiet room.

At this stage I had slight discomfort behind the sternum and when I bent over I could feel air bubbling from the apex to the base of my left lung. Next morning I went to the Sutherland Hospital where X-ray confirmed the presence of a small pneumothorax but did not show mediastinal air.

Fortunately I avoided an intocostal drain but spent 3 days in hospital and a week at home while the air absorbed. During this time I also developed a feeling as though I had some food caught in my oesophagus at the thoracic inlet. I presume this was due to air tracking in the mediastinum. At no stage did I develop subcutaneous emphysema.

After recovery from this incident I decided to investigate myself as fully as possible as being a keen diver. I wished to establish the risks involved in continuing the sport and to seek an explanation for the incident.

The first thing to do was to review the X-rays. I had had a full medical before commencing diving in early 1974. I obtained the chest ray from that time plus those taken in hospital and one after recovery. Radiological opinion suggested no abnormalities and certainly no obvious areas of overdistention or air trapping in any of the films.

The next step was to submit to a complete respiratory function work-up with Professor Colebatch at Prince Henry Hospital Respiratory Laboratory. This included total body plethysmography static and dynamic compliance testing with oesophageal balloons and determination of residual volume and airways resistance as well as the usual spirometry.

Professor Colebatch has recently published a paper[1] on Barotrauma in divers and was particularly keen to investigate. This work suggests that divers subject to barotrauma to the lungs constitute a population with decreased pulmonary compliance, possibly due to increased lung elastic tissue. He further suggests that this elastin is not uniformly distributed throughout the lungs and that this leads to relative

overdistention in those areas poor in elastin, driving ascent. He also feels that the air trapping hypothesis due to subpleural blebs or mechanical airways obstruction is an overworked hypothesis.

The results of these tests showed that I had a normal lung with very little tendency to fit his observations made on other divers with pulmonary barotrauma. There was no suggestion of air trapping or increased airways resistance. His advice was that it would be extremely unlikely that I would again suffer barotrauma should I continue to dive provided safe diving practices were followed.

Next I presented these findings to a thoracic surgeon familiar with diving problems and asked for an opinion as to the existence of subpleural blebs and as to the possibility that the incident might be a spontaneous pneumothorax unrelated to diving. He thought that the second possibility was very unlikely on the basis of my build and history of diving and also thought that air trapping in blebs would be unlikely.

Finally I consulted Dr Ian Unsworth of the Prince Henry Hyperbaric Unit. He agreed with the findings but advised that I should consider giving up diving as, although I may have normal lungs on all tests and a possible cause for barotrauma in the rapid 2 metre ascent mentioned above, the fact remains that I did develop a pneumothorax and that this probably increases the risk of further barotrauma in future. Air embolus is a more serious form of barotrauma that I may be subject to and if a rapid ascent were necessary in an emergency I would be at increased risk.

Armed with this knowledge and advice I have decided to cautiously continue diving but with the following self imposed changes in technique:

1. Only one ascent to the surface per dive.

2. Very slow ascent rates, at least half the recommended rate.

3. Attempt to maintain minimum lung volumes whilst diving and to avoid using lung volume as a method of buoyancy control.

4. Close attention to avoidance of glottis closure during diving.

5. Uniform use of buoyancy compensation vest and contents gauge to ensure surface flotation and adequate air supply on the bottom.

In addition I will in future dive with a buddy capable of resuscitating me and with facilities for tapping tension pneumothorax on the dive boat (ie. Cannula and Heimlich flutter valve).

I hope to report my future diving experience in this newsletter.

DR Kerr

1. MJ Colebatch et al. Increased elastie recoil as a determinant of Pulmonary Barotrauma in Divers. *Resp Physiol* 1976; 26: 55-64.

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Nothing’s for free

Mr Bell, British based managing director of Shell Oil Exploration said that his firm is now spending $1500 a minute on their North Sea oil operations.

A CLASSIC CASE OF DIVER AIR EMBOLISM AT THE SURFACE FROM HIGH WAVE ACTION

Howard W. Pollock*

On 25 September 1975 a 36 year old professional scuba diver and aquanaut with 17 years of diving experience, whom we shall call “Joe”, and who was a member of the First International Saturation Study of Herring and Hydroacoustics, died of an arterial air embolism to the heart and brain, as the apparent result of a surface re-entry accident. The accident occurred following a 3 day saturation dive (in the German underwater laboratory/habitat HELGOLAND in 110 feet of water, off Rockport, Massachusetts), and after a correct decompression phase, a proper instantaneous recompression for exiting the underwater laboratory/habitat, and a normal ascent, at least until the dive team was about 15' to 18' from the turbulent surface, where 10' to 12' waves were breaking against structures and equipment on the surface.

The three aquanauts were returning to the surface from the HELGOLAND via the buoy cable line attached to the relatively fixed and stable decompression buoy at surface. As observed from below, the decompression buoy extended about eight feet into the water below the surface in good weather conditions. But this day the weather conditions were not good, and the sea was not calm. Because of the pounding waves, the froth, foam and splash, the normally submerged portion of the stabilized decompression buoy was at one moment deeply submerged under the crest of a great wave and at the next moment nearly exposed above the surface as the trough of the wave would fall away from the base of the tethered buoy.

During the ascent up the decompression buoy line, all three divers exchanged several “OK” hand signals signifying that everything was under control and normal. This was done at the start of the ascent, at about midway, and again near the surface before exiting the water. One of the three divers later reported that he had likewise also exchanged “OK” signals with each of the other two divers immediately upon surfacing. Two of the divers surfaced in the heavy seas within a few feet of the decompression buoy, while Joe was first observed at the surface clinging to the buoy.

When the divers were rising to the surface together along the decompression buoy line, Joe was the first or uppermost diver on the line. When the last diver was about 10' from the submerged bottom of the fixed or stable decompression buoy, he and the diver just above him kicked away from the buoy and swam freely to the surface, bobbing like corks upon the crests and down into the troughs of the turbulent sea. Before swimming over to the pickup craft, one of the other divers looked back to see Joe clinging to the buoy with his Cressi full-face mask off and his mouthpiece (regulator) out of his mouth. He was apparently trying to attach a blue plastic bag to the decompression buoy, and was struggling with a Nikonos camera and strobe light at the same time.

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* Howard W Pollock is the Deputy Administrator of the National Oceanic and Atmospheric Administration and a former United States Congressman for Alaska. Mr Pollock is active in both national and international ocean affairs and is an experienced scuba diver who has vigorously supported increased efforts to tap the ocean’s resources and energy potential for the Nation’s benefit. He is the founder and Chairman Emeritus of the Congressional Underwater Explorers Club.

Finally he dropped the bag and camera and clung tightly to the fixed buoy staff with both hands as the heavy seas intermittently covered him. While he was relatively fixed in position, the waves were submerging him at crest and suddenly dropping away from him at trough. When he was next observed he was calling for help. He took a dunking and then cried out again in distress. A support diver reached him very quickly, hit the purge button of Joe's emergency (octopus) regulator to clear it of water, and immediately placed it back in Joe's mouth. At this juncture Joe relaxed and released his grip on the buoy. As he and the support diver slipped away from the buoy on the crest of a wave, the support diver fully inflated Joe's unisuit. He was then towed on his back by two support divers, unisuit inflated, regulator in his mouth, with his head out of the water.

Although he was virtually incapacitated on the return to the support vessel, there was an occasion when some of the seas washed over causing Joe's mouthpiece (regulator) to come out, and he personally replaced it with his right hand and held it in place momentarily: but, then both hand and mouthpiece fell away. One rescue diver later commented, "His eyes were open, although I can't recall seeing him blink as the water passed across his face." Another diver reported that when Joe was being towed a white frothy foam was bubbling from his mouth.

When he was placed aboard the support vessel, Joe showed no signs of life. He was pulseless and not breathing. Cardiopulmonary resuscitation was administered immediately and continuously aboard the support vessel as it proceeded directly to port. A Coast Guard helicopter was dispatched, but transfer of the casualty with the necessary life-support equipment in the rough sea conditions was determined to be unsafe. Upon arrival at Rockport Harbour, Joe was rushed to a recompression chamber by ambulance and was compressed to an equivalent depth of 165 feet (50 meters) of seawater for decompression treatment. After a time, when it was quite apparent that Joe was dead, the decompression phase and vigorous attempts at resuscitation were terminated. and the attending physician in charge officially pronounced him dead. Although a period of 3:19 hours elapsed from the time of the accident until the medical personnel on scene declared Joe dead and terminated resuscitation and recompression/ decompression treatment, it is probable that Joe was in fact dead within minutes of the embolism.

An autopsy confirmed the cause of death as an air embolism to the arteries of the heart and brain - presence of air forced into the cardiovascular system by changing pressure. More specifically, considerable quantities of air were found in the right and left ventricles of the heart, and air was present in all of the coronary arteries. In addition, an abundance of air bubbles was found in the vascular supply to the surface of the brain, and in the branches of the middle cerebral arteries. The embolism causes a sudden blocking of the artery by an obstructive air bubble which has been moved to its location in the vascular system by the blood flow. The tissues beyond the embolus become deprived of their blood supply, of course. Incidentally, it was especially noted that no gas could be forced out of the muscle tissue or subcutaneous fat by the application of great pressure or compression, which indicated that decompression had been quite complete prior to death.

According to the report of the Board of Investigation, the precise cause of the embolism could not be clearly established. The report indicated two reasonable possibilities:

a. "The high seas passing could have caused a momentary change of pressure sufficient to cause air embolism if the diver was holding fast to the mooring lines of the buoy and had just taken a full breath of compressed air.

b. “If the diver was rising to the surface with water in his mask and caught a relatively minute quantity of water in his larynx, this could produce laryngospasm with an associated constriction of air flow, or reflex breath-holding. Examination of the Cressi full-face mask on shore indicated that the right half of the face plate was out of its seal, leaving an opening over about 70% of the right side. There was also evidence of an impact on the top of the mask. It should be noted that this could have happened in the course of the rescue operations.”

The Board concluded that the actions which caused the embolism in the final ascent were those of the diver alone, and indicated that these actions were not what should have been expected of a diver of Joe’s long and distinguished record of diving under severe conditions. The Board questioned why Joe decided to surface at or on the buoy structure, for it was clearly hazardous because of the heavily surging sea and the attendant possibilities for impact or sudden changes in emersion.

One can only surmise from the available pieces of information precisely what happened to Joe. Not having been there, I can only attempt to reconstruct what might have occurred. Thus, as pure conjecture on my part, from a careful reading of the depositions, medical reports and supporting documents, and through personal discussions with other divers and support personnel involved in the incident, I am of the opinion that Joe must have received at least a glancing blow to the right side of his head near the temple as he attempted to surface, as a result of being thrown by wave action into the submerged portion of the decompression buoy. If he were dazed and disoriented this would account for his otherwise unexplainable actions at the buoy. It seems entirely logical that Joe’s full face mask with affixed regulator could possibly have been knocked away from his face. Accordingly, he certainly could have gulped a sufficient quantity of water to cause the laryngospasm with an associated breath-holding reflex. If this occurred at the precise moment when Joe was holding fast to the mooring lines of the buoy, and he had just taken a full breath of compressed air as the crest of a 10' or 12' wave fell completely out from under him, the momentary change of pressure in his lungs could have been sufficient to cause an air embolism. Obviously, this would be identical to a diver swimming up to the surface and holding his breath during the last 10' of the ascent.

Assuming nearly a one-half pound change in hydrostatic pressure for each foot of change in depth (actually it is 0.445 lbs change of pressure), there could have been about a five pound (250 mm Hg) immediate pressure differential in a 10' to 12' sudden change in wave height before Joe could expel the denser, compressed air which filled his lungs. Little more than 2 psi (pounds per square inch) or 100 mm Hg pressure differential would have been sufficient to force air into his circulatory system and cause the fatal embolism.

Perhaps it can be explained as follows. Air can be forced out from the alveoli of the lungs into the circulatory system when the air in the alveoli is at a higher pressure than that in the blood vessels surrounding the alveoli. Thus if a true pressure differential of about 100 mm Hg were to exist between the alveoli and surrounding capillaries, an embolism could occur; but there might be no clinical evidence of a rupture. 100 mm Hg pressure differential is equivalent to only 4 feet change in seawater depth, or only 2 psi of atmospheric pressure change. On the other hand, a true pressure differential of greater than 100 mm Hg could cause actual rupture of the alveolar capillary membranes.

The point is that such a critical pressure variation could have occurred each and every time a surging wave passed over Joe and then almost simultaneously fell away

below him as he held tightly to the buoy cable. If he inhaled a full lung capacity of compressed air at any such instant, a massive air embolism could surely occur. The same kind of danger of an air embolism can occur, of course, when a working diver attaches himself by magnets or otherwise to the side of a rolling ship, so that he is first immersed and then suddenly lifted above the surface when the sea drops out from under him.

The tragic lesson to be learned here is that a true pressure differential or change of only 4 feet of saltwater can cause an embolism, if one holds his or her breath in a maximum inhalation of compressed air, and a lung rupture might or might not occur in such a circumstance. Also, it is clear that a fatal air embolism can indeed occur to a diver after he reaches the surface if the necessary combination of circumstances conspire to occur at a common moment. If for any reason an emerging diver does not remain on the surface in turbulent wave conditions, and becomes alternately immersed and then lifted out of the water, it would be exceedingly important that only short, shallow and frequent breaths of compressed air be taken, so that the lungs are never filled to capacity and lose elasticity. Even though some elasticity remains in the lungs after a maximum inhalation, there is a danger of exceeding the elastic limit of the lungs and their alveoli if at this moment an expanding volume of air is introduced by a sudden decrease in depth and pressure. Also, it is important to emphasize another self-evident point, to wit: all divers should be cautioned to avoid fixed or stable structures when near the surface in rough weather. One other admonition bears mention. In heavy seas it is important to refrain from carrying objects if at all possible, so as to keep the hands free to use as necessary in any crisis situation.

In my view, this is a classic case of an air embolism occurring to a scuba diver as the result of a surface accident involving a fixed or stable structure in rough seas. It should be brought to the attention of all novice scuba divers as a part of their basic instruction. An understanding of the inherent dangers of diving in turbulent seas may save lives in the future. Somehow, I think Joe would have wanted it that way.

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Pots not natural habitat?

There is an interesting case simmering up in Newcastle at present. A man has been charged with stealing lobsters from another man's pots. As his defence he has claimed that the lobsters are the property of whoever first lifts them from the water, being still wild animals "free for the taking" until the owner lifted his pots. It was also claimed, naturally, that the State had no jurisdiction as the action occurred beyond the high water line. Legal evidence was offered to the effect that larceny could be committed on wild animals that are fit food for man. Sergeant Richards assured the Court that it is undisputed that lobsters are fit for human consumption. There is no mention of the lobsters pending the resumption of Court hearings ....

Cold gives little warning of the onset of Hypothermia. Abnormal behaviour (forgetfulness) may occur. 70% of the human body is within 2.5cms of the surface. Activity increases heat loss. Danger period continues after the victim has been removed from the water. Heat loss occurs even in "warm" water. Severe but reversible hypothermia may produce a death-like appearance and therapy be wrongly thought useless.

DECOMPRESSION SICKNESS IN DIVING

How, West and Edmonds

INTRODUCTION

Following the development of the air pump by Von Guericke in 1650, Robert Boyle was able to expose animals to decompression. In 1670 he reported these experiments and included the first description of decompression sickness - a bubble moving to and fro in the watery humour of the eye of the viper. Hoppe-Seyler repeated the experiments of Boyle, and in 1857 he described the obstruction of pulmonary vessels by bubbles and the inability of the heart to function adequately under those conditions. He also recommended recompression to remedy this. LeRoy de Mericourt in 1869, and Gal in 1872 described an occupational disease in sponge divers, which was also attributed to the breathing of compressed air and was equated with Caisson disease. Paul Bert in 1878 demonstrated in a most conclusive manner that the decompression sickness is primarily the result of an inert gas (nitrogen in the case of compressed air divers and caisson workers) which had been dissolved in the blood and tissues of the body, being released during or following the return to normal pressures. Gersh and Catchpole (1951) in reviewing the literature and their own work on the neurological manifestations of decompression sickness, demonstrated that gas bubbles formed in circulating blood after a short latent period from the time decompression commences. Most bubbles are filtered by the lungs; some pass through the lungs and reach the central nervous system and other organs and occlude arterioles of the same calibre. The clinical manifestations depend on the site of the vascular obstruction and collateral supply, and are largely a matter of chance. The early occurrence of venous bubbles often during the diver’s ascent and their subsequent emergence as a diminished quantity in the arterial system when decompression sickness develops, has been adequately verified by the ultrasonic doppler techniques in both animals and man.

Haldane, Boycott and Damant in 1907 demonstrated a technique of decompression whereby the diver ascends in a series of stages. This allows the gradual exhalation of the accumulated inert gas, thus reducing or preventing the number of bubbles able to form within the diver’s body. Recompression therapy was introduced for the treatment of decompression sickness, and this was modified by Goodman & Workman et al. (1965) with the introduction of 100% oxygen to hasten the elimination of inert gas from the body, while minimally compressing the diver in a recompression chamber to reduce the size of the gas bubbles causing the clinical symptoms. More recently the use of oxygen mixtures to bridge the compression gap between the air recompression therapy tables and the oxygen recompression therapy tables, allows greater flexibility in the treatment of this disorder (Edmonds et al., 1976). The very recent introduction of under-water oxygen recompression therapy has resulted in more rapid therapy being instituted in remote localities.

Clinical classification was presented (Golding et al. 1960) as an attempt to differentiate non-serious and serious cases, so that identification prognosis and therapy could be more appropriately standardised. This clinical classification of Type I and Type II decompression sickness is not wholly satisfactory. The classification was neither defined nor applied in the same spirit as it was initiated. Type I is defined as pain-only decompression sickness or joint bends. Type II includes those presenting with symptoms other than pain, or with abnormal physical signs. The central neurological, spinal, cardiovascular, respiratory and gastrointestinal manifestations are potentially serious. Naval (Bennett and Elliott 1969; Rivera 1963; Slark 1962) and recent Caisson series (Bennett and Elliott 1969; Griffiths 1969) have had a disproportionate dominance of joint bends, compared to the civilian series (Erde and Edmonds 1975). In the latter the neurological and cardiorespiratory

symptoms are much more frequent. To support or specify this variation in severity between the quoted Naval series and the rarely documented civilian cases, this relatively large series of civilian cases, are now examined. The only civilian series of comparable size was the Hawaiian group, described by Erde and Edmonds.

MATERIAL AND METHODS

A series of cases of decompression sickness were referred for treatment to two major recompression therapy centres in the Indo-Pacific region, the Singapore Armed Forces based at Singapore and the Royal Australian Navy School of Underwater Medicine at Sydney, Australia. The 115 consecutive cases included 40 from Singapore and 75 from Sydney. Records were made of the diving history, the clinical features, the response to treatment and other interrelated factors.

Inclusion of cases of decompression sickness in the series required either indisputable signs of this disorder or the development of clinical symptoms during or after ascent which were relieved or cured by recompression therapy. Cases of pulmonary barotrauma were excluded from the survey ie. those cases with haemoptysis, pleuritic pain, pneumothorax, mediastinal or cervical surgical emphysema, etc.

In assessing the result of treatment, the following grading system was used:

Complete cure 4 Almost complete cure 3
Definitely improved 2 No definite change 1
Clinically deteriorated or died 0.

The major treatment employed included the Air Tables 5A, 5B, 5C, 5D, 5E in the Australian Navy Diving Manual (these are equitable with tables 1, 2, 3 and 4 of the US Navy Diving Manual); the Oxygen Treatment Tables (6A, 6B of the Australian Navy Diving Manual) which are equitable with Tables 5 and 6 of the US Navy Manual; the high oxygen pressure tables, both in a compression chamber and underwater (Edmonds et al., 1976); and finally the use of oxygen at atmospheric pressure.

Symptomatic treatment was administered routinely, according to the severity of the case. Thus many received intravenous infusions, urinary catheterisation, steroids, anti-epileptic and tranquilliser drugs for neurological cases, as well as electrodiagnostic and clinical monitoring procedures.

RESULTS

Most of the cases were amateur or sports divers, fishermen divers, pearl divers, abalone divers or other locally employed divers. All were using compressed air as the medium, either in the form of self contained underwater breathing apparatus (SCUBA) or by the use of a line taking the air from a mechanical compressor on the surface or from cylinders also at sea level ie. a surface supply breathing apparatus (SSBA). Table I gives a description of the population involved and the type of dive profile.

As a generalisation, the dives were far in excess of those allowed by recompression tables. Only 24% attempted some form of decompression. As depicted in Table II, in 89% of dives there were sufficient data available to assess the decompression performed. The majority of both the Singapore and Australian groups exceeded the allowable duration underwater and did not perform adequate decompression staging. In 11% of cases there was insufficient information available to make a judgement in either direction. In those cases in which the dive was stated to be performed in accordance with the recommended tables, there is still some room for doubt as both depths and duration were often merely estimations.

TABLE I

	Mean	Standard Deviation	
Age	32.4	9.5	years
Depth	30.0	10.5	metres
Duration	120.6	112.1	minutes
Onset of Symptoms (from start of ascent)	33.1	48.0*	minutes
Delay in treatment (from start of symptoms)	50.9	40.4	hours

* not relevant due to extreme skew deviation

TABLE II

	Singapore	Australia	Total
Diver exceeded recommended tables	32 (80.0%)	55 (73%)	87 (76%)
Insufficient information available	1 (2.5%)	12 (16%)	13 (11%)
Dived in accordance with tables	7 (17.5%)	8 (11%)	15 (13%)
Total	**40**	**75**	

TABLE III

INCIDENCE OF SYMPTOMS N=115

Musculoskeletal	*71*	*(61.7%)*
Shoulder	56	48.7%
Elbows	42	36.5%
Arm	9	7.8%
Wrist	9	7.8%
Hand	1	.9%
Knee	21	18.3%
Hip	10	8.7%
Leg	9	7.8%
Thigh	7	6.1%
Ankle	3	2.6%
Neurological		
Cerebral	19	16.5%
Paresis and Paralysis	19	38.3%
Subjective sensory loss	59	51.3%
Loss of sensation	54	47.0%
Loss of proprioception	21	18.2%
Loss of bladder function	41	35.6%
Loss of bowel function	21	18.2%
Pain in spinal column	15	13.0%
Unconsciousness	23	20.0%
Inner Ear		
Vertigo	10	8.7%
Deafness	3	2.6%
Tinnitus	2	1.7%
Respiratory		
Chest pain	18	15.6%
Cough	12	10.4%
Dyspnoea	20	17.4%

Gastrointestinal		
Abdominal pain	18	15.7%
Nausea	25	21.7%
Generalised		
Malaise	31	27.0%
Dizziness	20	17.4%
Anorexia	6	5.2%
Fever	4	3.5%
Integumental		
Oedema	10	8.7%
Itching	9	7.8%
Rashes	6	5.2%

Table III gives the overall incidence of symptom attributable to decompression sickness, and includes all those symptoms or signs which are recorded in the case reports. It does not in any way attempt to infer severity.

Table IV shows the predominant manifestations, this is described as either a Type I or Type II decompression sickness. Type I is usually designated as minor manifestation of decompression sickness, and Type II as a serious manifestation.

TABLE IV

PREDOMINANT DECOMPRESSION SICKNESS MANIFESTATIONS

Type I - Decompression Sickness	54 cases	47%
Type II - Decompression Sickness	61 cases	53%
Cerebral	11 cases	10%
Spinal	22 cases	19%
Both spinal and cerebral	22 cases	19%
Inner ear	3 cases	3%
Cardiorespiratory	3 cases	3%

Type I Decompression Sickness

This is the less serious form of decompression sickness as regards morbidity and mortality, mainly affects the musculoskeletal system, and this is commonly termed “Bends”. Of the total, 47% fell into this category, although 15% more cases had evidence of joint pains as well as more serious symptoms, thus placing them into the Type II decompression sickness group. As in most other series of decompression sickness affecting divers, the upper limbs were most affected, with the following order of predominance - shoulders, elbows, knees and hips. As in a previous series, when multiple joints were involved, they tend to occur in neighbouring joints.

Type II Decompression Sickness

In the Singapore cases, spinal lesions dominated the clinical presentation. The Australian cases had a greater number of cerebral and cardiorespiratory manifestations, probably reflecting the closer proximity of the diving to the therapeutic facility. It was noted that cerebral and cardio-respiratory manifestations tend to occur soon after the completion of the dive, and in some cases resolve spontaneously. Sometimes spinal lesions supervene after a delay. Spinal cases are much less correctable by

procrastination, and therefore are more likely to be transported long distances, taking a longer time. Aborted or inadequate water recompression therapy was often attempted and usually served to aggravate clinical symptoms and result in unnecessary delay of treatment.

In the case of Type II decompression sickness there are often other manifestations, which can be seen from Table III. Spinal cord involvement was the most likely cause of severe persistent disability from decompression sickness.

Times of Onset of Symptoms.

Although the mean time between ascent and the first presentation of symptoms is 33.1 minutes, a standard deviation is not relevant as the distribution is strongly skewed. Twelve developed symptoms during the repetitive dive, with ascent or whilst staging. Thirty-one cases developed symptoms upon surfacing or very soon after. A further twenty-three within the first ten minutes making a total of sixty-six decompression sickness cases developing within this time. The longest duration between ascent and the initial development of symptoms was 19 hours, and this particular case did respond well to recompression therapy. The second longest case developed symptoms 5 hours after ascent.

TABLE V

FREQUENCY OF RESPONSE TO THERAPY

Response (grade)	Singapore		Australia		Total
4	19	49%	52	72%	63%
3	12	31%	11	15%	20%
2	4	10%	5	7%	8%
1	4	10%	2	3%	6%
0	0	0	2	3%	2%

The comparison of the responses to treatment from different therapeutic regimes is a rough one, and definite conclusions cannot be drawn from this comparison, as the cases were not randomly selected for each regime. On the contrary, the severity of the case and the local conditions were paramount determining factors in the decision as to which therapeutic regime to apply. Thus oxygen would be administered “on the surface” at 1 ATA in a mild case of Type I decompression sickness of a long duration and distant from recompression facilities. Originally in the Singapore group there was a tendency to treat according to the Air Tables, although in later years the Workman’s Oxygen Tables were applied. The Australian group were more often treated with the Australian high oxygen pressure Tables ie. the maximum safe oxygen pressure administered either in a compression chamber, underwater or at 1 ATA.

In assessing the response to therapy the previous mentioned grading system is used:

Complete cure	4;	Almost complete cure	3;
Definite improvement	2;	No definite change	1;
Clinically deteriorated or Died	0.		

It is important to realise that the response is compared to the initial severity of the case.

The total number of regimes used exceeds the number of cases, because some cases responded insufficiently to one type and so required another. In the majority of

cases oxygen was also used intermittently following the recompression regime, to avoid or diminish the recurrence of minor symptoms. In many of the paraplegic cases ie. those with spinal decompression sickness, hyperbaric oxygenation was also used subsequently to the initial recompression therapy. This regime is used both in the Singapore and Australian areas with apparently good clinical results.

When observing the time required for full recovery, it was observed that 60% were fully recovered within 24 hours of initiating treatment. Another 17% recovered within one month, 14% more within the year. Approximately 7% were permanently affected, and 2% died from the decompression sickness.

TABLE VI

RESPONSE TO VARIOUS TREATMENTS

	Mean Response	Number
Air Tables	2.29	24
Workmen's 0_2 Tables	2.31	59
Australian 0_2 Tables	3.58	52
0_2 administered at 1 ATA	3.2	10

DISCUSSION

The more serious nature of civilian cases, as compared to the Naval series was again evident. The dramatic superiority of Workman's Oxygen Tables over the conventional Air Tables was not as evident as in the Naval series. Perhaps this was because of the more established and serious nature of the civilian cases. This in turn may be due to the specific problems encountered amongst a non-disciplined diving population. These problems include:

- Divers who are physically unsuited for the type of dive they are performing eg. due to obesity, etc.
- The unawareness of correct diving procedures, eg. decompression staging, dive planning, etc.
- Using inadequate equipment, often without depth gauges or underwater watches for decompression.
- No facilities for decompression. Insufficient gas supplies, etc.
- The performance of rapid ascents, there being frequently a history of an emergency or unplanned ascent.
- Unnecessary delays in early treatment.
- The local administrative authority being unprepared for diving accidents, aggravating the delays.
- Inadequate local recompression treatment facilities.

The advantages of Workman's Oxygen Tables to the operators were very evident requiring less time and resulting in less inconvenience to the therapists. Edmonds' oxygen underwater treatment regime was of use in remote localities, when the divers were prepared for the eventuality of decompression sickness. In this regime the patient is submerged with a supply of oxygen from the surface, comprising a large (220 cubic foot) oxygen cylinder with the hose extending to a maximum depth of 9 metres underwater, and having a demand valve attached. The demand valve fits through a full face mask, thus reducing the dangers of unconsciousness, vomiting underwater and drowning. A companion diver is required, but because of the depth there is no problem regarding decompression sickness in the assistants. The diver sits on a stage or

a weighted shot rope hanging over the side of the boat. If possible a sheltered lagoon or harbour is preferred to reduce the incidence of sea sickness in both the diver and the attendant. After 30-120 minutes the diver is brought to the surface at a rate of 12 minutes per metre, by hauling the shot rope upwards. The diver continues breathing oxygen throughout and is left on intermittent oxygen (one hour on, one hour off) when he reaches the surface to reduce the incidence of recurrence in most cases. This regime will usually result in a considerable relief or complete cure, but even at the worst the diver is given some hours of nitrogen elimination, a reduction of the severity of the clinical complications, and time in which transport can be arranged to the nearest recompression facility.

When transport is obtained it is imperative that it is achieved with as little physical disturbances as possible, and carried out at a pressure equivalent to sea level. Both the physical disturbance and the exposure to lower than atmospheric pressure will increase the volume of gas bubbles associated with decompression sickness. Many civilian and commercial aircraft can be pressurised to 1 ATA and are thus suitable for the trip for diving casualties. During transportation humidified oxygen may be administered to the patient. All other treatment procedures are based on general medical principles. The blood volume deficit should be remedied, cerebral and spinal oedema reduced, haematological aberrations remedied, and specific symptoms (convulsions, delirium, urine retention, gastrointestinal symptoms, etc.) must be treated on their merits.

In experience of both authors, delayed cases are often treated with hyperbaric oxygen, as opposed to the attempts to remove the symptoms by increased pressure. This is especially so in cases of spinal decompression sickness of some days duration. Despite this, an attempt should always be made to remove or reduce symptoms initially by the application of pressure, and to remove bubbles by the inhalation of the highest oxygen mixture possible, with reference to oxygen toxicity. These are more fully described in the conventional medical texts on diving (Bennett and Elliott, 1969; Edmonds et al., 1976).

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WHY WORRY ABOUT DROWNING? *
Col. Richard H Wood

We may be approaching this problem of drowning (or not drowning) all wrong! We confuse the cause of death with the cause of the accident - and they *aren't* the same.

When someone falls, we know it's the sudden stop that really smarts - but we don't spend much time on that end of the problem. Why, then, don't we accept drowning as the expected result of trying to breathe water and concentrate on preventing the situation that causes it? *"Don't we?"* No. Not to any great extent. The thrust of most water safety programs is aimed at the idea that better swimmers, more lifeguards, and closer supervision will prevent drowning. To a certain extent, they will; but when you consider that drowning is the second largest cause of accidental death the Air Force, you'd have to say that the total program has some weak spots.

One weakness is the emphasis on rescue. We are, in effect, putting a mattress under the building instead of a railing around the balcony. Another weakness is that we naturally assume that a good swimmer won't drown. Since good swimmers *do* drown, there may be something about drowning that is not related to swimming proficiency that we are overlooking.

Let's try a little prevention. Since drowning will occur whenever a person tries to breathe while his mouth and nose are immersed in liquid, let's speculate on what might cause that.

For openers, we can sav that anyone who loses consciousness in the water is a likely candidate for drowning. He hasn't much choice. That suggests that the running, head-first dive may not be our greatest athletic achievement. It's hard to think of any other activity where we so willingly use our head to scout for obstructions. But most people who drown are not *initially* unconscious. Furthermore, they know how to swim. They may even be good swimmers. *Why do they drown?*

Accident reports aren't much help. Just when the report approaches that moment of truth, that instant when we are finally going to learn the secret of drowning, it gives us a verbal shrug of the shoulders:

"He became exhausted."
"He exceeded his limitations."
"He drowned."

There are some new ideas about drowning that make a lot of sense and suggest a new approach to prevention. Dr Michael B Strauss describes what is probably the leading cause of drowning. It goes like this: a problem (currents, fatigue, surf, leg cramp, cold water) causes the victim to panic. The victim begins breathing very rapidly, and the exchange of oxygen and carbon dioxide becomes increasingly inefficient. The rapid breathing results in shallow breathing and is extremely exhausting. The relatively small amount of air kept in the lungs may cause the victim to become less buoyant, which requires more effort, which increases the breathing rate and the panic .... this "vicious circle" may lead to the collapse of the victim. Once he collapses, drowning becomes a formality.

Keep the above description in mind for a minute while you read the report of an actual drowning. The following investigation was unusually complete because the victim had

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been scuba diving, and his “buddy” made an excellent witness.

“ .... they proceeded toward the exit point, about 50 yards away. Before reaching the exit point, swimming with the current, X began to tire and ‘acted scared’. The current carried them beyond their exit point. They began to look for a suitable exit (the shoreline was a rocky cliff) but found none. X was tired, breathing very hard, and becoming more frightened. As X’s breathing became more laboured, he became more frightened, and his buddy felt it imperative to get him out of the water as soon as possible. They began an exit over the ledge at the base of the cliffs, but a wave loosened their grasp and pulled them back into the water. At this point, X quietly drowned.” Just like that! He quietly drowned!

I have a high degree of confidence in that description. Over a few years’ worth of teaching scuba diving, I have watched, on several occasions, a supposedly good swimmer go from a relatively controlled situation to a state of near collapse with frightening speed - sometimes in as little as 30 seconds.

They all start with rapid breathing. The victim is spending energy, perhaps because he’s cold, frightened, over-exerting himself, or a combination of these; and his respiratory system is trying to keep up by demanding more air. To keep breathing rapidly, he must keep his mouth and nose fully clear of the water and this requires even more energy, which demands more rapid breathing, and so on. Viewed underwater, his arm and leg movements become faster and less efficient, which requires even faster movement and more energy. Back on the surface, breathing rate is accelerated at a runaway pace. The process appears irreversible, and collapse is seconds away. Without assistance drowning is inevitable.

“Aha,” you say, “if I become exhausted in the water, I’ll merely float on my back and rest while I recover.”

“Aha,” I answer. “I don’t think you will, and I’ll tell you why.”

To begin with, most of us float only if we control our breathing, keep our chest expanded, and keep a certain amount of air in our lungs. A person gasping for breath does not do any of these. Next, many swimmers can float only if they relax and assume the floating position they were taught. An exhausted swimmer gasping for breath is anything but relaxed. Third, if there is any wave action at all, it takes a certain amount of energy to keep from being tipped over or swamped by each wave. Ocean floating isn’t quite as easy as pool floating. Finally, cold (or cool) water is a user of energy all by itself. Even if you manage to relax and float without movement, the body’s answer to being cold is to spend energy.

The point of all this is that once you become exhausted in the water, you can’t simply stop and do nothing while you catch your breath. You must spend a certain amount of energy just to stay afloat and breathe. If you are spending it faster than you are recovering it, you are going to collapse and drown.

Now think for a moment about your own personal reaction to exhaustion. Can you approach exhaustion and then recover from it by merely slowing down? Up to a point, you probably can - but beyond that point you’ll have to stop. And that’s when swimmers become drowners.

In testing applicants for Scuba training, I’ve tried a number of different swimming tests. Finding out who can swim is simple enough, but finding out who won’t drown is another matter. The test I like best, at this writing, is to require applicants to swim far enough (440 yards) to get good and tired and then go immediately into

20 minutes of survival swimming without any rest. I don't care whether they float, tread, bob up and down - I want to see them recover from exhaustion while avoiding drowning. You might be surprised at the number of good swimmers who find that difficult, but that's what it's going to be like in the real ocean. You just can't stop and hang onto the side of the ocean while you catch your breath.

Let me sell you on this one point. Rapid breathing in the water is a *real* danger signal. It tells you that your "energy" system is out of balance and is going to zero if you don't Stop it. Regardless of how good a swimmer I may be, I know that if my breathing is rapid *and increasing,* I am getting close to my own point-of-no-return. I must stop and recover before I get to that point.

Now, that's not easy. A swimmer does not become exhausted for no reason. There is an objective involved. Perhaps the swimmer is trying to get to a boat or to the shore. Perhaps the boat is drifting away and he is trying to catch it. Recognizing exhaustion and stopping to recover is *not* what the swimmer wants to do. He is, literally, like the guy who drives faster so he'll get home before he runs out of gas! He is usually willing to spend all of his energy in pursuit of his objective; and he is rarely in a mood to listen to reason. Failure to reach his goal may be inconvenient or even dangerous, but failure to stop and recover from exhaustion can be fatal!

So, recognize exhaustion for the danger that it is and decide right now that you are going to handle the exhaustion problem first and any other problem next.

Secondly, if you are assisting an exhausted swimmer, don't be in a big hurry to get him out of the water if it is going to cost him energy. Get the exhaustion under control first. Unburden him (if he is burdened); give him something to hang onto or support him so he won't have to spend any of his own energy to stay afloat; and help him relax while he recovers his breath and his composure. Getting him out of the water is not an essential part of the procedure.

* * * * * * * *

The paper by Dr Kerr is an extremely interesting account of Pulmonary Barotrauma from the customer's point of view. If you couple this with the account from Howard Pollock, which has been delayed in presentation by a lack of space in previous issues, you will perhaps suspect that an attempt is being made to start you thinking about the critical factors concerned in causing symptomatic pulmonary barotrauma. There are too few reports readily available and case notes are requested, or at least note of the existence of cases. Understanding of this disease is at present inadequate yet is critical for a medical Ex Cathedra ruling on the practicing of "Free Ascent" as a training procedure.

The above may help you gain the maximum benefit from the articles that follow, to whose writers sincere thanks are given. These originate in this issue from Australia, Singapore, the USA and the UK. To misquote, "Underwater is underwater is underwater" the whole world round, and the fullest sharing of information and hard won experience possible is highly desirable. It seems pointless to die to prove that we can make the same mistakes as the other fellow.

It is appropriate at this time to offer hope for a prosperous, happy and disaster-free New Year. In this respect the cartoon, and we all owe a debt of gratitude to Mr Peter Harrigan will serve better than a picture of Santa.

* * * * * * * *

A CAVE DIVING INCIDENT - A LIFE IN THE BALANCE *
RS Dickens

Somewhere along the line of a well planned cave diving training program, the subject of rescue and recovery is introduced. Unfortunately, the emphasis must be placed on recovery rather than rescue, because until a short time ago, only one successful cave diving rescue had been made (to the knowledge of this author). The dramatic rescue was made by Sam DiPerna and a team of his divers in the Peacock Slough cave which is located near Branford, Florida. Sam is a member of the PADI Board of Advisors for Cave Diving, a PADI Master Instructor, and a PADI and an NACD cave diving instructor.

Upon completion of a swim through from Olson to Peacock, Sam DiPerna and his team of cave divers were resting on the surface of Peacock when they were told that divers from South Carolina had entered the Peacock cave improperly equipped for cave diving. Sam, uneasy because of the report, questioned the instructor/group leader of the diving party and learned that not one of the divers was cave qualified. Sam and members of his team re-entered Peacock and began a search for the divers at once. They passed through the slit, entered the cave, and found the divers approximately 150 feet into the cave from the bottom of the slit. The divers started swimming toward the slit as soon as they saw Sam's lights (one was walking on the bottom, pulling himself along by using the guide line).

Just prior to ascending the slit, one diver suddenly removed his mask, spat out his regulator and pulled off his buddy's mask. The two divers danced around, stirring up a great amount of silt. By the time Sam reached them, they had unsuccessfully attempted manual buddy breathing. The victim was hanging onto the line from underneath with both hands and was wearing no mask, had his eyes closed, and was holding his breath. Sam placed his octopus regulator in the victim's mouth, and the victim began to breathe. However, he was totally immobile, and would not leave the line to ascend (at this point the line ran vertically up the slit). The intensity of the distressed diver's fear was evidenced by Sam's inability to pry his fingers from the line.

In an effort to calm the man, Sam tried to replace the mask, but was unable to do so. He then backed up the slit and began an attempt to pull the diver out by his valve. The man released the octopus rather than be pulled out, and refused to take the octopus again. Sam made a second attempt to pull the diver out, but could not hold him.

No longer able to hold his breath, the victim released the line and settled to the bottom. His feet were entangled in the line. Two members of Sam's team cut the line and delivered the victim to Sam. The victim's weight belt was removed while Sam tilted the head back to allow the expanding air to escape. Sam then commenced a seventy-foot ascent to the surface, at an angle of about forty degrees, with the unconscious victim.

When Sam reached the surface, he pulled the victim over to a ledge and removed his tank. A grey-brown foam began to spew from his mouth, and later turned to pink. He was cyanotic and unconscious. Sam directed the initiation of mouth to mouth resuscitation, and the victim was lifted to the bank feet up. His colour was still cyanotic and there was no carotid pulse or breathing. The eyes were totally dilated. Sam then commenced CPR, and calls to the sheriff were made from a nearby gas station. By the time the sheriff arrived, the victim's pulse had been re-established. He was trying to rise, but was kept in first aid for air embolism position-head down, feet

up, and on his left side. Rescue from Live-Oak arrived, administered oxygen, and received instructions from Sam to transport the victim in the embolism position to the nearest chamber which is in Gainesville Florida.

Sam interviewed the victim's partners and examined the gear and reported the following:

1. The victim was 18 years old and had just recently been certified, but had no cave training.
2. He was over weighted - over 20 pounds of lead.
3. His tank still held 400 psi of air, and the regulator functioned properly on the surface.
4. The "J" valve on his single 71.2 cubic foot tank had no pull rod.
5. His submersible pressure gauge came off his regulator at an angle that did not allow his "J" valve mechanism to operate - the "J" hit the hose and could not be activated.
6. His BC had no CO_2 mechanism or any other way to inject air into the vest except orally, and the vest was tied on.

Following are a number of observations made by Sam about the accident:

a. Although there were a number of persons at the site, no help was offered to the rescue team. At the time the team was administering CPR, some members of South Carolina group were still suiting up to go into the water.
b. The rescue took only six minutes from the time Sam and his team submerged until they surfaced.

In a review of the event, Sam made the following analysis of procedures:

1. W*hy wasn't the line cut earlier to free the diver's legs?* Cutting a guide line is such an alien concept to a cave diver that it is almost outside consideration. If the victim's "security blanket" had been removed, he might have gone completely berserk.
2. *Could the victim's BC have been inflated and his weight belt dropped earlier?* The BC had no CO_2 inflation, and in a confined space, extra buoyancy might have been a handicap. In a cave the only place to go is to the ceiling - there is no surface.
3. *Oxygen should have been given, but Sam's team had none.* They will carry it in future.
4. According to Sam, the South Carolina leader and his party made these errors:
   a. The students were not warned about the dangers of cave diving.
   b. There was insufficient control of the group.
   c. Inexperienced divers should never have been taken to such an area.
   d. Many members of the party seemed unfamiliar with emergency procedures for air embolism, CPR, etc.
   e. Their equipment was not properly assembled.
   f. They did not have proper training nor equipment for the dive they made.

The victim did not embolize. He recovered from an ugly accident only because an experienced team of certified cave divers happened to be near enough to rescue him.

Cave diving can be a safe and rewarding activity if done with respect for rules and training, but negligence and ignorance most often lead to a tragedy rather than an incident. To Sam DiPerna, who tipped the balance in favour of life - WELL DONE.

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DIVING SAFETY MEMOS -
UK Department of Energy

Through the courtesy of Commander SA Warner, Senior Inspector of Diving for the Petroleum Production Directorate, copies of this service to the diving profession have been made available. They cover the problems that have been noted to occur in relation to Diving Bells (SDCs), masks, both Scuba and hose supply diving, HPO and electrical risks and give a suggested basic medical kit for both SDC and DDC use. They even comment on the fact that putting a DDC on the deck may alter the stability of the diving boat. The comment concerning the need for an efficient hoist in the bell is to noted especially as in a recent Australian case the victim could only be partially brought into the SDC, this preventing closure of the door, limiting the decompression procedure, delaying raising of the victim and interfering with resuscitative procedures. The memos are intentionally brief, being intended to alert diving contractors and divers to matters requiring more attention. They are a valuable service and greatly to be commended. The following is a brief resume of points made:

Diving Bells should only be operated from a suitably stable platform, having regard to the sea and weather record of the area. The resistant drag experienced when starting to raise the bell and its weights from the sea floor can create considerable problems. NO absolutely safe design for underslung ballast which would be safe in all types of sea bed conditions has yet been designed. When using underslung ballast technique the bottom door of the bell should always be shut before the weights are broken from the sea bed. Lifting harness should give a pelvic lift, NOT shoulder or chest (Memo 2 3 8 14).

Scuba Except in conditions where the use of surface supply diving equipment makes the divers task impossible or more hazardous, the use of Scuba is not recommended (at oil rigs). In two recent fatalities the first stage reducer provided air to both the demand valve and the suit inflation connection, a matter being investigated for its significance (Memos 9 11).

Hose-supply problems include the fouling and rupture of the diver’s umbilical. In one case the diver, who was aware of the fouling, had removed his mask at the surface but was then pulled back into the sea from the diving basket by the hose. There is a warning that design inadequacy of the system may allow the “bail out” bottle to vent if the supply hose is ruptured. Similarly, the free flowing air may so reduce available pressure that the stand-by diver cannot obtain an adequate supply. Both the individual diver’s gas reserve and the emergency diver supply MUST be separate from the main gas supply of the diver (Memos 10 14).

Masks have been noted to have defective fittings on occasion (Memo 6).

Cathodic Protection for divers is noted as necessary (Memo 12).

ire Risk from HPO is noted when opening up cylinders of HPO into unpressurised or “contaminated” lines. The production of PHOSGENE from the over heating of PTFE is another risk noted (Memo 4).

Basic Medical Equipment to be maintained at all times with a bell (SDC) or DIMZ is listed in Memo 7.

These Memos are designed to “alert” professional divers and diving contractors to problems that have arisen to trouble, sometimes fatally, diving operations in the North Sea in association with Oil Rigs and pipes. They naturally draw on experience from other areas also but the chief of the killing grounds for divers appears to be

the Oil Rigs. The problems of the North Sea include multi-nationalities (among companies, divers and languages), cold water, undersupervision and undertraining of divers who have a great money stimulus to work often beyond their abilities, and a "Gold Rush" atmosphere. Caution and moderation are somewhat at a discount, and "necessity" the spur in such an environment. The deaths that have occurred, concerning which too little information is available, appear to be due to the usual factors, viz ignorance, greed, carelessness and diving that is "beyond the state of the art" at the site. Such opinions, it should be needless to add, flow from other sources of information and are NOT to be ascribed to either Commander Warner or the Department of Energy.

Once again the need for full Incident Reporting is noted as a SAFETY measure to reduce future mortality and morbidity. Failure to heed such calls will result in needless suffering and costly compensation claims. The latter consideration should move if mere appeals on grounds of humanity are disregarded! It is unlikely that diving in the South Pacific area will be conducted any more carefully than that in European waters, or that the lack of morbidity reports indicates a higher standard of practice. This is an appeal for the more open discussion of all serious diving-related problems. The legal liability risks will be less in the long, or even short, term from seeking to remedy problems than from trying to deny them. This is something that can only come about if ALL parties involved are agreed that safety and efficiency are ultimately in harmony.

"WE LOST A DIVER, BUT THE DCM IS FINE"

SPUMS - Oceans Society Meeting at Frankston, Saturday 4 December 1976, starting at 1.30 pm

The Oceans Society of Australia exists to further our knowledge of the oceans of the world and the seas surrounding our island continent. They organise the successful Oceans meetings at Monash University on the Queens Birthday Weekend each June. When they heard about the proposed SPUMS meeting in Frankston they offered to do the administration and help SPUMS run the meeting. Without their help and hard work the meeting would have been much less successful.

The venue was the George Jenkins Theatre at the State College of Victoria in Frankston. A magnificent new and very comfortable theatre suitable for plays, lectures and films. The cost of the hall and the services of the projectionists made it necessary to charge admission. Undesirable but unfortunately very necessary.

The Proceedings were opened by Dr Bill Rehfisch, the Treasurer of SPUMS, outlining the ideas behind the meeting. In his opinion knowledge was the key to diver safety and here were knowledgeable doctors to talk about topics that affect every diver. He also gave a plug for the two organising societies.

Then a film on the mechanical effects of pressure, made by the USN in the 1960s was shown. While mostly accurate it has some “deliberate (?) mistakes”, which were pointed out by the first speaker, Surgeon Commander Geoff Bayliss. He spoke on aural and sinus barotrauma and then showed some X-rays of pulmonary barotrauma and in describing the circumstances of the patients made it quite clear that free ascent training is a dangerous game.

The audience asked some very intelligent questions and the panel offered answers that seemed to satisfy. Then another film was shown. This time an RN film made in 1973, entitled “Decompression Sickness” This was a little confusing as it was metric and the USN film was in feet and psi whereas the plummy pommie voice spoke about metres and bars. However the message came across clearly. By the end of the film the audience were suffering from too much sitting so the tea break was taken. Then Dr John Knight spoke about cold and the diver and nitrogen narcosis and then he spoke on decompression sickness, the symptoms presenting when the tables are followed closely (as in USN diving) and the very different incidence of symptoms when decompression is taken lightly. He then discussed how to avoid decompression sickness.

Dr Chris Lowry discussed diving safety and drowning pointing out that death in the water was seldom due to regulator failure, but often due to dangerous diving habits. There was an interesting discussion about the right sort of life jacket buoyancy compensator. The general opinion of the meeting was that not one brand on the market met all the requirements.

The meeting closed with the RN film “Give Him Air” where we were introduced to Joe and Josie and the various ways they could die from lack of air, summarised as “not air”, “airway blockage”, “chest injury” and “stab wound”. The last was illustrated with a spear gun accident when the spear pierced the chest and most realistic blood bubbled out. The first aid treatment was depicted and anyone who has seen the film should be able to cope with an unconscious patient and give mouth to mouth resuscitation, or as the film called it, “The Kiss of Life”.

continued on page 30

BOOK REVIEW

"Diving Medicine"
RH Strauss, MD

(1976, Grune & Stratton, 111 Fifth Ave, New York. US $21.00)

This American text on Diving Medicine by 21 authors is well presented and easy to read. 17 of the authors are resident in the USA, 2 in Canada, one in the UK (Professor Denis Walder) and one in Australia (Dr Carl Edmonds). It is unfortunate that the first chapter, on the history of diving and diving medicine, perpetuates a myth. Augustus Siebe did not invent the diving dress and helmet. This was first patented by John and Charles Deane and it was this equipment that was used in the successful salvage of guns from and the destruction, as a hazard to shipping, of the "Royal George" in Spithead.

On the whole the information supplied is accurate, and considering the authors this is to be expected, but there are some surprises. Few authorities would accept a PCO_2 of 50 mm Hg as normal (p 149). CO_2 convulsions are hardly a risk to divers, the unconsciousness that will supervene earlier is a much greater hazard.

The chapter on ear and sinus problems in diving gives an excellent overview seen from the viewpoint of the ENT consultant to an academic hyperbaric unit. But the recommended treatment for external ear barotrauma can hardly be expected to comfort the patient. The section on paranasal sinus barotrauma makes it quite clear that the authors have little personal experience with this common diving problem. The suggested pathology is that of absorption of air and vacuum formation, yet at the same time exudation of tissue fluid. The explanation offered in "Diving and Subaquatic Medicine" by Edmonds, Lowry and Pennefather, of raised external pressure compressing the gas trapped in the sinus and leading to exudation and rupture of the sinus mucosa, is easier to understand and to explain to affected divers.

There are a number of references to unpublished observations and personal communications. This is an extreme form of academic one-up-man-ship as such references are unverifiable. On page 193 there is a reference number 41 but there is no 41 in the list of references. Pages 404 and 405 and 406 and 407 are numbered the right order but the contents of pages 406 and 407 should come before those of 404 and 405.

The chapter on drugs and diving is page filling waffle and contributes nothing. Neither does the chapter on diving accidents. Most of the information is rehashed in the chapter on the investigation of diving accidents. In spite of the grumbles above the book it is worth buying and reading. After each chapter there are simple questions to test the reader's comprehension and the correct answers are at the back of the book. Here also are to be found 10 case histories, the last being a damning indictment of doctors who dare to care for divers without being adequately trained. Misdiagnosis killed the diver.

The USN standard air decompression tables are incorporated but the instructions for

their use are omitted. Prof AA Buhlmann's decompression tables for diving at altitude are included. These are the only well tested altitude tables available at present. Unfortunately again the instructions have been omitted.

To sum up: Diving Medicine by Strauss gives a wide-ranging overview, written by acknowledged experts, of diving medicine. It is however weak on practical diving medicine as we see it in Australia and on its clinical aspects. For this the reader would have to turn to "Diving and Subaquatic Medicine", Edmonds, Lowry and Pennefather which, though less easy to read, has much more clinical meat for the reader to feed upon.

John Knight.

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After the meeting the Oceans Society put on some wine and biscuits in the foyer so that small group discussions could occur and these were later carried on by the organisers at Bill Rehfisch's home with an excellent supper.

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ACKNOWLEDGEMENT

A Cave Diving Incident - A Life in the Balance
RS Dickens

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